THE

AMERICAN REVIEW OF TUBERCULOSIS ABSTRACTS

VOLUME XLVIII SEPTEMBER, 1943 ABST. NO. 3

Epidemic Virus Pneumonia.—During the months of February and March, an epidemic of virus pneumonia was observed in a battalion of sharpshooters. The disease was characterized by malaise, high and moderately high temperatures, slight cough and expectoration, which was bloody in only one case, and defervescence by lysis. Objectively 59 out of 105 patients in whom the diagnosis was made showed X-ray changes. These consisted of infiltrations of variable extent and density, not confined to one lobe. The physical signs were scant. The sputum did not show a predominating organism. A leucopenia was present early in the course, later on a moderate leucocytosis occurred. The disease pursued a benign course. There were no fatalities. Three times pleural effusions complicated the course. Haemorrhagic tendencies were observed three times, namely epistaxis, muscle haematoma and bloody sputum. The disease was highly contagious within the group of soldiers exposed but showed no tendency to spread to the civilian population.—*Influenza-artige Epidemie mit gehäuften Lungeninfiltraten (Viruspneumonie) in einem Füs. Bat., E. Haemig & W. Heyden, Schweiz. med. Wchnschr., October 10, 1942, 72: 1113.*—(*H. M.*)

Influenzal Pneumonia.—A small epidemic of influenza is described occurring in a school of adolescents. In all there were 28 cases during the period. In one group of about 10 cases the symptoms were very slight and lasted a few days only. Of the remainder, 12 showed a bizarre course. The temperature at the start in this latter group was seldom more than 102°F., remaining so for one to two days; it then dropped to between 99° and 100°F., and remained there from three to seven days. The symptoms consisted of a mild headache or a sore throat. Apart from the temperature the only positive finding was a pharyngitis. After a week or less the temperature became normal and the symptoms cleared up. After a further period of two to seven days, however, the temperature suddenly rose again, in one or two cases quite steeply, and remained above normal for some days, in a few instances for two weeks or even longer. During this period a cough often appeared, a few very faint crackling râles were heard over a localized area of one lung. Seven of these cases were radiographed and in all cases with one exception, a typical roentgenogram was found: an area of one lung-field was distinctly blurred—frequently the lower third of the right lung-field—the movement of the corresponding dome of the diaphragm was impaired (fluoroscopically), and in one case there was a small effusion in the costophrenic angle. Later control roentgenograms revealed that this diffuse shadow had disappeared and the lung-field showed no abnormality. In 2 cases, however, a recrudescence occurred after an interval of four to six weeks, with exactly the same symptoms, and repeated films showed that the previous shadows had become more marked. All cases recovered completely—in one case sulphapyridine was used but the course was the same as in untreated cases. The authors conclude that: (*1*) the course of the illness is not typical of an influenzal

pneumonia; it might belong to another group of virus diseases; (*2*) as no other cases have been noted in the district among adults, it may be that the illness only attacks adolescents; (*3*) owing to the mildness of the illness and the absence of any marked symptoms, it will rarely lead to hospital treatment and its special characteristics and complications may easily be overlooked; the attention of the practitioner should therefore be drawn to it.—*Atypical "Influenzal Pneumonia" at a School, H. G. Herxheimer & A. J. McMillan, Brit. M. J., October 31, 1942, 2: 513.*—(*D. H. C.*)

Pulmonary Atelectasis.—Previous animal experiments of the authors have demonstrated a striking reactivity of the bronchial tree to the stimulus of pulmonary embolism. It is their feeling that the initiation of bronchial obstruction may be due to early bronchomotor and bronchosecretory phenomena which may subsequently result in mechanical obstruction. Their experiments have shown that bilateral vagal section inhibits bronchospasm and that intravenous atropine sulfate, particularly with the addition of papaverine, prevents its occurrence in 60 per cent of the experiments. Associated with a reflex bronchoconstriction, an increased bronchial secretion occurs in the experimental animal. These two essential factors, bronchoconstriction and increased secretion, invariably induced by experimentally produced pulmonary embolism, frequently result in atelectasis. Stimuli other than pulmonary embolism may affect the bronchial tree equally. The authors have demonstrated that intraäbdominal manipulations, such as pulling on the mesentery, also trauma to the chest wall with or without rib injury, were capable of changing the calibre and pattern of the bronchi, the effect of these stimuli also being modified by the use of atropine. The authors now have data on 28 patients suffering from pulmonary embolism, who received atropine-papaverine medication for three days, and in none of which cases was there any development of massive atelectasis. Traumatic atelectasis deserves more recognition than hitherto. Roentgen studies of patients with chest wall injuries showed some evidence of pulmonary change in three-fourths of the cases, indicating a nervous reflex causative mechanism in these cases. It has also been demonstrated that injections of iodized poppyseed oil may occasionally produce bronchial spasm followed by atelectasis. The early recognition and adequate treatment of reflex atelectasis is stressed by the authors.—*Reflex Pulmonary Atelectasis, G. de Takats, G. K. Fenn & E. L. Jenkinson, J. A. M. A., October 31, 1942, 120: 686.*—(*G. L. L.*)

Linear Shadows in Lung.—A combined roentgenological and pathological study was made of cases showing linear and band-shaped shadows in the lungs. These shadows were found to be produced by interlobar pleurisy, plate-shaped foci of atelectasis, healing or healed infarcts and the scarring of old destructive infections. The clinical histories in cases of interlobar pleurisy, infarct, atelectasis and scarring in the lungs are often similar enough to make it impossible to use clinical data alone in differential diagnosis. In addition, there is an intimate histological and pathological relationship. For example, an infarct may produce pleurisy, atelectasis and scarring, pleurisy may produce atelectasis and interlobar scarring; persistent atelectasis may become infected secondarily and these subsequent inflammatory changes may produce pleurisy or fibrosis. Interlobar pleuritis and plate-like atelectasis are much more common than healed infarcts. If the anatomical location of the linear shadows corresponds to the position of the interlobar fissure, the interlobar pleurisy is readily differentiated from healed infarcts or plate-shaped atelectasis. The pleurisy may present as a long line of equal thickness corresponding to the length of the fissure but it is usually associated with other evidence of pleurisy such as fluid or pleural thickening near it. Although infarcts frequently are adjacent to the interlobar pleura, they are seen as a more nodular uni-

laterally placed shadow near the thin line of the readily recognized interlobar fissure. Plate-like atelectasis does not occur in the location of interlobar fissures. If the linear shadow is perpendicular to one of the interlobar fissures, interlobar pleurisy is readily ruled out and the differential diagnosis lies between healed infarct and plate-shaped atelectasis. Healed infarcts and plate-like atelectasis, although often difficult to differentiate, may show the following features: the shadows of infarcts are usually shorter than atelectasis; they are in a single plane, each infarct producing one line; they may end in nodular extremities and may run in any direction. The plate-like foci of atelectasis are usually long, traverse the entire portion of the lung involved, extending from one pleural surface to another; they are often multiple, occur in different planes, one focus producing multiple closely placed nearly parallel lines and they show no nodular irregularities. In atelectasis the pleura may show a V-shaped retraction but there is usually no regional thickening. Other important differences between these two lesions are: (*1*) the linear shadow of atelectasis usually disappears within a few days, whereas that of a healed infarct persists; (*2*) the diaphragmatic excursion is nearly always limited in plate-like atelectasis, but this feature is rare in a healed infarct; (*3*) although both atelectasis and infarction occur predominantly in the lower portions of the lungs, infarcts unlike atelectasis are not uncommon in the upper lung fields.—*Linear Shadows in the Lung, F. Fleischner, A. O. Hampton & B. Castleman, Am. J. Roentgenol., November, 1941, 46: 610.*—(*W. H. M.*)

Bronchocolic Fistula.—Bronchocolic fistula may be produced by direct perforation of an abscess (empyema or subphrenic abscess). The course which the pus follows after perforation of the diaphragm depends on whether or not a free pleural space exists. If the basal pleura is not adherent, perforation is followed by an extensive empyema, which may later rupture into the bronchus. In the presence of adhesions, the abscess will rupture into the bronchus, or a small, encapsulated empyema may be formed, subsequently to rupture into the bronchus. The length of time between the evident onset of symptoms produced by the original focus of infection and the establishment of a bronchocolic fistula is difficult to determine without the study of a large series of cases. The clinical diagnosis of a bronchocolic fistula may offer great difficulties. This complication should be considered whenever, in the course of an empyema or subphrenic abscess, the patient develops a sudden severe pain, cough, and expectorates moderate or copious amounts of foul sputum, without clinical or roentgenological evidence of a definite pulmonary abscess. The examination of the sputum may demonstrate the presence of a definite pulmonary abscess. The examination of the sputum may demonstrate the presence of *B. coli*, and the feces may contain frank pus. A roentgenological demonstration of the fistula should be attempted. Bronchography may prove an adequate procedure if the lipiodol should visualize the fistulous tract and empty into the colon. If this method fails to confirm the clinical diagnosis of a bronchocolic fistula, a barium enema should be given. These fistulae may be of a valve type, and it is difficult to predict which of these methods may prove more successful. In the author's case, a subphrenic abscess following an acute appendicitis and perforative appendicitis apparently ruptured simultaneously into a left lower lobe bronchus and the distal transverse colon. During the prolonged course of the disease, the patient developed a chronic pneumonitis at the left base and an encapsulated empyema.—*Bronchocolic Fistula, A. J. Ackermann, Am. J. Roentgenol., February, 1942, 47: 294.*—(*W. H. M.*)

Irradiation Fibrosis of Lung.—A clinical and roentgenologic study was made of the chests of 273 patients receiving radiotherapy for cancer of the breast. Sixty-two, or 22 per cent of the 273 cases, showed roentgenographic evidence of fibrosis. The fibrosis

with atelectasis was a permanent change and appeared within two to twelve months in varying degrees after each of three or four skin portals had received doses of 1600 r to 2000 r in a single cycle of therapy or doses of 3000 r to 6000 r in two or more cycles (r measurements in air). In only 9 of the 62 affected cases, or 3.3 per cent of the whole series of 273, was there no evidence of complicating metastatic cancer of the lungs after being observed from three to ten years. The other 53, or 85 per cent of the affected cases, were shown subsequently to be complicated by metastatic cancer. The author feels it is justifiable to infer that, when fibrosis of the lungs occurs after irradiation, there is an underlying contributory factor which seems to be metastatic cancer, (85 per cent of affected cases) or possible infection, arteriosclerosis or advancing age (15 per cent of affected cases or 3.3 per cent of whole series). There was no resulting physical disability from the fibrosis. Cough, pain and dyspnoea occurred to very slight degrees, but when these symptoms were marked and associated with failing health and strength, the eventual termination of the case was proved to be the result of metastatic cancer. Roentgenological determinations of irradiation fibrosis complicating and accentuating metastatic cancer or other possible factors of pathology are fraught with many complexities and inconsistencies, so that interpretations and conclusions may be controversial. Standards of evaluation are only approximate estimations in dealing with a clinical subject.—*Irradiation Pulmonary Fibrosis, B. P. Widmann, Am. J. Roentgenol., January, 1942, 47: 24.*—(*W. H. M.*)

Irradiation Fibrosis of Lung.—In the treatment of cancer of the breast in part or entirely by high voltage roentgen therapy, permanent pulmonary fibrosis is a major complication. Of 347 cases treated between 1926 and 1936 and who are still alive, 22.1 per cent (77 cases) developed roentgenographic evidence of pulmonary fibrosis. Different radiotherapeutic methods were employed and had to be evaluated. The frequency of irradiation fibrosis is directly related to the large total doses delivered to the lung by divided dose technique and the volume of lung tissue irradiated. In this series of cases, there is no evidence that age is a predisposing factor. Although elderly people are believed more prone to develop pulmonary fibrosis, some of the most severe cases were observed in persons in the fourth decade. To date, no fully adequate pathological study of the postirradiation changes in human lungs has appeared. On clinical and pathological evidence, it is known that pleuropulmonitis may be a transient reaction in lungs and may disappear, leaving no demonstrable residue. On the other hand, it may not subside but persist, eventually going on to produce permanent fibrotic lesions. The chief symptoms are persistent, slightly productive cough, dyspnoea on exertion and fatigue. Haemoptysis, pain and palpitation are less common. Classically, deviation of the trachea, retraction of the mediastinum to the affected side, diminished thoracic expansion, impaired percussion note and high-pitched tubular breath notes are easily recognized. Less intense forms of the disease often escape detection, although they may cause marked distress to the patient. In general the greater the amount of radiation delivered, the more severe the late pulmonary symptoms are likely to be, but outstanding exceptions are so frequent that one cannot make it a clinical axiom. Not one instance of chronic *cor pulmonale* in patients with uncomplicated irradiation fibrosis has been observed. However, when the mediastinum is markedly displaced and fibrotic, right heart failure may occur not because of the pulmonary scarring, but because of angulation of the pulmonary vessels with resultant right ventricular strain, hypertrophy and finally dilatation. Consistent electrocardiographic changes are observed very frequently but it is now known that these are not the result of primary or secondary cardiac disease but are due to alterations in the relationship of the heart to other structures in the chest. The chronic cough is explained

by persistent chronic bronchitis in the affected area. Based on the studies of the alterations of pulmonary physiology in these patients the dyspnoea is due to: (*a*) marked diminution of the vital capacity caused by the fibrosis itself; (*b*) a variable degree of fixation of the chest wall; (*c*) compensatory emphysema of the uninvolved portions of the lungs; (*d*) heightened irritability of the inspiratory and expiratory pulmonary reflexes of Hering and Breuer, thus seriously altering the normal rhythmic action of the entire sequence of pulmonary reflexes. The dyspnoea may be relieved by means of an abdominal belt or weight reduction or both. The cough improves as the dyspnoea decreases.—*Fibrosis of the Lung following Roentgen Irradiation for Cancer of the Breast, J. E. Leach, J. H. Farrow, F. W. Foote, Jr. & N. W. Wawro, Am. J. Roentgenol., May, 1942, 47: 740.*—(*W. H. M.*)

Apical Scars.—A group of about 50 specimens of apical scars was examined. Causes of death were chiefly cancer and late cardiovascular disease; tuberculosis played no part in the illness or death of these patients. The typical scar was observed to include a pleural lesion capping one or more secondary lobules of the lung and also an underlying parenchymal lesion corresponding to the pleural lesion. These lesions varied in extent and degree but there was little variation in their fundamental features. The pleura shows an increase in connective tissue of varying density; this tissue is vacularized, even when very dense and is apparently the result of organization of fibrinous exudate at some period. In most cases there is an increase in width of the fibres of the elastica propria. The parenchymal lesion may be superficial or extend as a wedge shaped scar to a depth of over a centimeter. It consists essentially of a necrosis of the lung framework with obliteration of blood supply and intraälveolar exudate organized into vascular connective tissue; the latter varies in density and cellularity depending on age of the scar. Alveolar and bronchiolar walls are well marked out by elastic tissue, revealing variable degrees of alveolar collapse but not complete atelectasis. Lymphoid infiltration is relatively common and pigment-bearing phagocytes are also present. Tubercles have been found only in the deep wedge shaped scars. Numerous unsolved questions are presented by the study. Apical scars may result from sudden necrosis of a sensitized portion of the lung following a second infection with tubercle bacilli followed by flooding of the alveoli with serous exudate which is eventually organized. In the deeper scars the tubercles lying near the apex may be older than the scar and responsible for the tissue sensitivity.—*The Pulmonary Apical Scar: An Inquiry, C. H. Bunting, Arch. Path., July, 1942, 34: 67.*—(*D. G. F.*)

Surgical Risk in Asthma.—In order to check the mortality risk and the incidence of postoperative pulmonary complications, the authors present a study of 189 consecutive cases of allergic asthma and asthmatic bronchitis who received major surgery at the Mayo Clinic between 1934 and 1940. A total of 25 (13.2 per cent) patients developed some form of pulmonary complication (including severe asthma) following operation. Of these, 5.3 per cent developed postoperative pneumonia, 4.8 per cent severe asthma, 1.6 per cent bronchitis, 1.1 per cent atelectasis and 0.5 per cent pulmonary infarction. The incidence of complications in patients with a preoperative diagnosis of allergic asthma was only slightly greater than in the cases of asthmatic bronchitis. Patients with asthma and asthmatic bronchitis who undergo operations on the upper parts of the abdomen, however, are more likely to have postoperative pulmonary complications, including exacerbations of severe asthma, than those who undergo operations on the lower part of the abdomen. Generally speaking, the risk of operation on these patients is not great if they receive adequate preoperative and postoperative care, since 86.7 per cent of the series developed no significant postoperative pulmonary complications.—*Is the Asthmatic Patient a Good Surgical Risk?, F. W. Gaarde,*

L. E. Prickman & H. J. Raszkowski, J. A. M. A., October 10, 1942, 120: 431.—(*G. L. L.*)

Asthma and Histamine Therapy.—The vegetative nervous system is known to play an important rôle in the production of asthma. The rôle of histamine in its genesis is not entirely clear. The similarity of cutaneous lesions produced by histamine and those seen in urticaria has suggested that histamine may be responsible for the nonspecific tissue response of allergic conditions, including asthma. Several theories regarding its production in the lung during an asthmatic crisis have been proposed. It is assumed to be produced endogenously by the action of the autonomic nervous system or its local production may act as an irritant to the autonomic nerve endings. The presence of histaminase in the normal lung would support such an assumption. The author has treated 105 cases of asthma by desensitization treatment with histamine iontophoresis. A small amount of 2 per cent histamine ointment is applied to the skin of the back, and a current of 5 milliamperes is applied for from three to five minutes depending on the local reaction. The number of treatments that is necessary to produce improvement is variable. Frequently a dramatic response to a few treatments is obtained, and in other cases up to thirty treatments must be given. Of the author's cases, 84 per cent were improved and over half the cases, 54 in all, were cured of their asthmatic attacks.—*Asthme et histaminothérapie, A. Dudan, Schweiz. med. Wchnschr., July 25, 1942, 72: 821.*—(*H. M.*)

Cor Pulmonale in Asthma.—The incidence of cardiac complications arising after prolonged bronchial asthma was studied in 15 cases of bronchial asthma that came to autopsy and an additional 54 living cases. The effect of bronchial asthma on the right side of the heart is difficult to estimate clinically. The right-sided hypertrophy is seldom great enough to make itself evident by roentgenographic study. The most common and important clinical feature is a tendency to right axis deviation and other electrocardiographic changes indicative of right ventricular strain and myocardial damage. The diagnosis of congestive heart failure is often made difficult by dyspnoea of emphysematous origin. Five of 12 autopsied cases with bronchial asthma of six or more years' duration died in congestive failure and 4 of these showed predominant hypertrophy of the right ventricle.—*The Occurrence of Cor Pulmonale in Cases of Bronchial Asthma, I. W. Schiller, A. Colmes & D. Davis, New England J. Med., January 28, 1943, 228: 113.*—(*J. B.*)

Heart in Emphysema.—Twenty-eight patients with emphysema of various types and grades were subjected to contrast visualization of the cardiac chambers and great vessels following the injection of 70 per cent diodrast solution. These patients were unselected. Most were ambulatory. The technique of injection was that described by Robb and Steinberg. Recording of the roentgen findings was carried out by various methods. In all cases visual observation and multiple photographs of the fluorographic screen were made on 35 mm. film by the fluorographic method. In many cases these were supplemented by moving pictures of the fluoroscopic screen, roentgen kymograms and direct roentgenograms. Clinical history, physical examination, posteroanterior roentgenograms, electrocardiograms, circulation studies and vital capacity were recorded in all patients. In 24 of the 28 cases there was some evidence of right ventricular dilatation or dilatation of the base of the pulmonary artery. In 50 per cent of these, or 43 per cent of the total, dilatation of the right ventricular cavity was unequivocal and the interventricular septum was convex to the left (in normal individuals the septum is convex to the right). In this rather small series, right axis deviation or interventricular block of the type described by Wilson was constantly associated with abnormalities of the right cardiac chambers. The normal electrocardiogram did not preclude the findings of right ventricular dilatation. On the contrary in 8 cases, well defined dilatation

with deviation of the septum was present. The lack of right axis deviation in some of these cases might be accounted for by the coincidental presence of left ventricular enlargement. In 2 cases of marked emphysema with chronic bronchitis of long duration, there was no right ventricular dilatation. No explanation is offered for this finding. In 2 other cases in which a normal right heart was found the clinical symptoms and normal vital capacity suggested extremely mild involvement. Coincidental left ventricular dilatation could be accounted for in many instances by essential hypertension or previous coronary thrombosis. In the remaining cases coronary sclerosis seemed to be the most likely explanation. In the presence of right ventricular dilatation there was also an abnormality in the movement of the interventricular septum. Analysis of this phenomenon has not been carried out sufficiently at this time to permit its explanation.—*Contrast Visualization of the Heart and Great Vessels in Emphysema, M. L. Sussman, M. F. Steinberg & A. Grishman, Am. J. Roentgenol., March, 1942, 47: 368.*—(*W. H. M.*)

Mediastinal Emphysema.—Thirteen cases of spontaneous mediastinal emphysema have been published up to the present time. The diagnosis can usually be made without any difficulty. A twenty-two year old man was admitted to Bellevue Hospital with severe pain in the left hemithorax. The pain had started one hour prior to the admission when the patient was sitting quietly in a chair. On physical examination there was subcutaneous emphysema over the left chest. The percussion note over the precordium was unusually resonant. Over the left chest "boiler-like" sounds were heard, which disappeared within two hours. A roentgen examination revealed a small pneumothorax at the apex of the left lung. The white cell count was 19,500, with 93 per cent polymorphonuclears and 7 per cent lymphocytes. The crunching sound heard over the left side of the chest is supposed to be a characteristic finding in mediastinal emphysema and makes easy its differentiation from coronary occlusion, pericarditis, ruptured or dissecting aneurysm and pulmonary embolism.—*Spontaneous Mediastinal Emphysema, R. M. Lintz, Arch. Int. Med., February, 1943, 71: 256.*—(*G. C. L.*)

Pneumonoconiosis in Talc Industry.—Fifteen cases of pneumonoconiosis in talc workers are reported with necropsy studies in 5. Analysis of the dust to which the workers were exposed indicates that the offending agents are two silicates, tremolite and soapstone. Free silica was not found in any of the samples. The symptoms are those characteristic of disabling pneumonoconiosis: dyspnoea, chest pain, cough and weakness. Cyanosis, clubbing of the fingers and very low vital capacity are present. The gross pulmonary changes common to all cases are stiffening of the lungs, parenchymal bands and nodules, and moderate enlargement of the tracheobronchial lymph nodes. Emphysema is universally present. All advanced cases without important complications exhibit *cor pulmonale*. Microscopical changes consist chiefly of irregular areas and bands of fibrosis surrounding the vessels and bronchi, producing thickening of the alveolar walls. Histiocytes and fibroblasts are numerous. Occasional foreign-body giant cells and dust laden macrophages are present. There are moderately numerous small areas of necrosis and, where alveoli are occluded by fibrosis, there is hypertrophy of epithelium and occasional cell masses simulating carcinoma. An interesting finding common to all cases is the frequent presence of asbestosis bodies which implies a degree of similarity between asbestosis and pneumonoconiosis due to talc. The chemical relation of the two dusts is apparent, both being silicates. Although the predominant particles in crushed talc are small crystals, there are spicules and fibres. It has yet to be determined whether all of the fibrosing effect of the silicates is due to the presence of fibres. Diffuse haziness is the most commonly noted roentgen finding and is probably the earliest detectable change. This may be accompanied by a background of a fine in-

definite reticulation of lines. The appearance suggests that the diffuse haziness due to interstitial fibrosis has been modified by a fine emphysema. Another manifestation is nodulation. The nodules are less dense and less well defined than those of typical silicosis. They vary in size and tend to become confluent. The distribution is usually generalized but may be of greatest intensity in the parahilar or subapical regions or near the bases. Confluence of lesions may indicate a comparatively advanced stage. The formation of conglomerate masses may be evidence of associated infection but this supposition has not received clinical or histological confirmation in any of the cases. The shadows of the hila are often large and dense. Severity of symptoms appears to be more definitely related to the type rather than to the extent of the lesion seen roentgenographically. Moderately extensive coarse nodulation may cause only mild discomfort, such as dyspnoea after physical exertion. Fine diffuse haziness is more disabling. If uncomplicated, talc pneumonoconiosis is so unlike the usual type of pulmonary tuberculosis in symptomatology and in the roentgenographic distribution of lesions as to make differential diagnosis comparatively easy. Even where tuberculosis is established as a complicating factor, the principle cause of disability is often pneumonoconiosis. In uncomplicated cases, the onset of symptoms marks the beginning of a degree of disability from which there may be no recovery. Workers should be withdrawn from exposure prior to this time. Roentgen changes are present before the onset of dyspnoea or other subjective symptoms. The determination of vital capacity may be an effectual routine procedure in selecting workers that should be more thoroughly studied as potential subjects for withdrawal from further exposure. Standard exercise tests for the objective period determination of dyspnoea may aid in the discovery of early pulmonary fibrosis. Measures to reduce the dust concentration should be instituted.—*Pneumoconiosis in the Talc Industry, F. W. Porro, J. R. Patton & A. A. Hobbs, Jr., Am. J. Roentgenol., April, 1942, 47: 507.*—(*W. H. M.*)

Lung-Dusting Experiments.—Using cats as subjects, various radiopaque dusts (two kinds of finely powdered lead glass, bismuth carbonate and uncompressed gas flame carbon) were introduced into the lungs by different methods: (*1*) direct insufflation, (*2*) inhalation during quiet respiration, (*3*) inhalation during spasmodic respiration. Control lateral and posteroanterior roentgenograms of the thorax were taken before and immediately after the dustings. The lungs and trachea were immediately exsected and roentgenographed. Additional roentgenograms were taken after fixation of the lungs and the removal of portions for histological examination in order to locate the exsected portions in the previous films. All of the animals were found to have dust within the alveoli. Although the amount of alveolar dusting varied much, it was never so great as to be detectable with certainty in the roentgenograms, even of the exsected lungs. This was in marked contrast to the obvious bronchial dusting. The amount of alveolar dusting did not run parallel with the amount of bronchiolar and bronchial dusting. Almost identical quantities of a particular dust could be present in the alveoli of two subjects, while the bronchioles and bronchi of one were loaded and the other practically clear. The authors feel that, regardless of the method used in the experiments, the amount of dust in the alveoli is about the same. More experimental data concerning this are required for confirmation. Introduced dusts appear to collect in relatively greater amounts in inflamed portions of lungs. In a number of cases alveolar dust cells were found to have ingested dust particles within a matter of minutes from the penetration of these latter into alveoli. There was no roentgenological evidence of any deposition of dust in the bronchial lymph nodes, although some of the subjects had received several insufflations of radiopaque dust over a period of many weeks. It is therefore felt that, normally, the loaded alveolar dust cells move toward the ciliated

epithelium of the bronchioles, are caught up on the layer of viscid mucus, and are carried up with it by the action of cilia. (The absence of accumulations of radiopaque dust in the lymph nodes seems to indicate that this route of excretion is little used in the case of healthy animals.) The dust brought up into the pharynx is swallowed and passed out by the alimentary tract (demonstrated roentgenographically in earlier experiments). When the inhalation experiments were modified by the intratracheal injections of a few cubic centimeters of fluid, alveoli were rendered radiopaque over large areas of lung and the excretion of the dust was considerably delayed. The slowness of excretion is ascribed primarily to the actual quantity of dust carried down by the fluid and to the consequent overwhelming of the protective dust cell mechanism which is not adapted to meeting demands of this order. These findings suggest that excessive watery secretion from the bronchiolar and bronchial glands, such as might occur in humans under certain pathological conditions, would tend to carry dust down into the alveoli. An outstanding feature in the experiments was the apparent existence of a defense mechanism preventing the entry of large quantities of dust into alveoli, for the total quantities in them were comparatively small, even when the bronchioles were heavily loaded.—*Further Lung-Dusting Experiments, A. E. Barclay, K. J. Franklin & M. M. L. Prichard, Am. J. Roentgenol., March, 1942, 47: 362.*—(*W. H. M.*)

Silicosis.—Early roentgen findings consist of increased density of the hilum and prominence of linear markings but these are also found in many conditions other than silicosis. Discrete nodulation is the one essential finding required to establish the diagnosis of silicosis; the nodules are small and rounded, vary from 2 to 6 mm. in diameter and have sharply defined borders; they are first seen near the hilum, usually on the right side, and later spread uniformly and discretely throughout both lung fields though less prominent at the bases and at the periphery. Once present, silicotic nodules never disappear. Coalescent nodulation occurs when the nodules become more numerous in a fairly small area and the fibrous tissue between them becomes more evident; these areas are most commonly seen in the central portion of the upper lung fields and are almost always bilateral and symmetrically placed. Conglomerate masses are usually due to the merging of a number of these coalescent areas with the addition of fibrous tissue, into a dense area of larger size; usually regular in outline, they may at times appear quite irregular. They, too, are usually in the central and upper part of the lung fields, though they sometimes occupy the greater part of the upper two-thirds of one or both sides. Emphysema is quite common in the older and more advanced cases; it begins usually in the costophrenic angles and extends to involve the lower one-fourth or one-third of the thoracic cage; it is usually bilateral and symmetrical. Fibrous bands are common in advanced cases, they extend from the dense fibrous conglomerate mass in the upper lung area and pass through the emphysematous region causing tenting of the diaphragm. The acute or rapidly developing type of silicosis, which is rare, is most frequently seen in the young adult employed in an alkaline or almost pure fine silica dust atmosphere; roentgen examination is of little help in diagnosing this type of silicosis, as the disease may occur and be disabling within two or three years after the initial exposure to the dust, too early for the nodulation to form and be shown; the roentgenograms appear hazy or fogged with only a moderate increased density in the hilum. At postmortem the lungs contain gross numbers of microscopical discrete nodules and have lost much of their elasticity with definite increase of fibrous tissue. *Cor pulmonale* is seen only when the elasticity of the lung tissue is almost nil, or in advanced cases of fibrosis and conglomerate masses with its associated high-grade emphysema. Pulmonary tuberculosis is more common and the mortality from it is greater in the silicotic than the general population; cavities in the lungs may occur in the silicotic lung totally

free of tuberculous infection. In the differential diagnosis in the earlier or moderately advanced cases of silicosis, conditions to be considered are miliary tuberculosis, siderosis, the small metastatic malignant deposits especially from hypernephroma, many of the yeast fungi deposits, Boeck's sarcoid and sprue. In the more advanced cases, conditions to be considered in the differential diagnosis are pulmonary tuberculosis, cancer, emphysema, bronchiectasis, atelectasis and the lung changes which naturally occur in advancing age (when not only is there more fibrous tissue but the lymphatics themselves are normally more evident), and chronic cardiac disease with the usual accompanying arteriosclerosis and lung changes due to the decompensation.—*The Value of the Roentgen Ray in the Diagnosis and Prognosis of Silicosis, W. M. Doughty, Am. J. Roentgenol., November, 1941, 46: 605.*—(*W. H. M.*)

Bagassosis.—Bagasse is the term applied to broken sugar-cane after the sugar has been extracted. This formerly waste product of the sugar-cane industry is now employed in the manufacture of board. The bagasse arrives at the board-making factory in open bales, which are then "broken"—that is, the raw material is further crushed into small fragments. Originally this latter process was carried on under water—which is a slow process. Recently, because of war demands, it is carried out in the open—giving rise to a great deal of dust particles, some very finely divided, in the air near the machinery. Bagasse, which is largely composed of fibre, contains about 1 per cent protein and 5 to 7 per cent silica. Some of its cellulose content is said to be present in a crystalline form. Directly after the installation of the dry bale-breaking machinery, cases of respiratory illness began to appear among the workers engaged in this part of the process. No cases of respiratory trouble had previously occurred in the factory. Four cases of this new condition—termed bagassosis—are described. The first case is that of a nineteen year old laborer with negative previous and family history. After one month's exposure he noted increasing shortness of breath and cough associated with scanty sputum occasionally blood-streaked. He was admitted to the hospital about six weeks after onset of symptoms and was found to be dyspnoeic at rest and cyanotic. The temperature was slightly elevated and roentgenogram on admission revealed lesions suggesting bilateral bronchopneumonia of influenzal type. After two days of slightly increased temperature he remained afebrile for the remainder of his course. Cyanosis with extreme dyspnoea persisted for three weeks then both gradually subsided over the next four weeks. A film two weeks after admission showed an increase in the areas of consolidation. Sulphapyridine had no influence on the course of the disease but nasal oxygen relieved the dyspnoea. Signs of patchy consolidation persisted in both lower lobes for seven and one-half weeks, with râles frequently in other areas. Finally the dyspnoea gradually ceased, the chest began to clear two months after admission, and the patient became symptom-free, with resolution of the lung lesions three and one-half months after initial hospitalization. The sputum was always scanty, mucopurulent and at times blood-streaked. No definite organism could be isolated. There was a slight leucocytosis which promptly returned to normal. Three other very similar cases are described—in 2 of them some residual fibrosis was seen on the chest film after symptomatic recovery. The clinical appearance of these patients suggests an acute inflammatory lung disease with urgent and extreme dyspnoea as the presenting symptom but with little or no febrile reaction. Various diagnoses such as bronchopneumonia, tuberculosis and chronic pneumonia were entertained originally in these cases but the circumstantial evidence of exposure to the same dust as well as the peculiar clinical picture made the authors ascribe these cases to bagassosis—an industrial lung disease. It would appear that exposure to bagasse dust is capable of initiating a pathological process in the lungs, the complete resolution of which is possible with a return to normal,

or which may progress to a lesion the nature of which is probably fibrotic such as occurred in 2 of the 4 cases described—in both of which greater and longer exposure to the dust was present. All 4 cases were found sensitive to an antigen of the bagasse dust—whereas controls were negative. Further, it was noted that when the protein was removed from the antigenic material no sensitivity resulted. The authors conclude that the acute phase of bagassosis is possibly an allergic response in the lungs to this antigen. The pathological process might be due to: (*1*) a form of silicosis which supervenes upon the allergic phase during or after the latter's resolution; (*2*) a response on the part of the lungs to crystalline cellulose or (*3*) a chronic process of fibrotic nature occurring in tissues oedematous from their allergic response to the antigen.—*Bagassosis: An Industrial Lung Disease, L. I. M. Castleden & J. L. Hamilton-Paterson, Brit. M. J., October 24, 1942, 2: 478.*—(*D. H. C.*)

Bagassosis.—The authors' attention had been called to the lung condition designated bagassosis by a previous report (Castleden and Hamilton-Paterson). They saw 4 cases, 2 of which had been described in the previous report. Their interest was aroused, first when one of the men related a series of symptoms which did not fit into any well known clinical picture and, second, when he remarked that several other men who had worked on his machine, a "bagasse-shredder," had shown identical symptoms. They then investigated the others. They note the following outstanding features which appeared to characterize this condition: (*1*) an "incubation period" of exposure to dust of from two to four months duration; (*2*) sudden onset of the acute phase; (*3*) the particularly violent and continuous cough; (*4*) the type of sputum which was variously described as dark, green, blue, dirty, like manure, with a burning taste; (*5*) retrosternal pain aggravated by movement; (*6*) great weakness; and (*7*) mental depression, loss of weight and appetite. Physical examination revealed, in addition to previously noted findings, that exercise tolerance still remained decidedly poor in 3 of the men, long after the acute phase had passed. Bronchoscopic examination revealed: (*1*) abnormal congestion of the trachea and bronchi; (*2*) excessive frothy mucus; (*3*) glazing of the mucosae. The legal aspect is discussed in view of the fact that no payment under the Workmen's Compensation Act could apparently be claimed, as the disease is not a scheduled one.—*Bagassosis: Further Notes of Four Cases, J. A. Gillison & F. Taylor, Brit. M. J., November 14, 1942, 2: 577.*—(*D. H. C.*)

Lung in Azotaemia.—A diagnosis of azotaemia can be made from a roentgenogram of the chest. The pulmonary changes occur, however, in only a small percentage of patients having nitrogen retention. The degree of elevation of blood nitrogen does not parallel the extent of lung changes; marked pulmonary findings may occur with only slight elevation of blood urea. From a roentgenographic viewpoint, it seems that the pulmonary changes associated with the azotaemic state are simply an exaggeration of those occurring in acute pulmonary oedema without a nephritic background. It is most probably related to left ventricular failure. The roentgen appearance is that of a symmetrical, diffusely increased and irregular density located in the central and basal lung fields. The density decreases gradually as it extends to the lung periphery. The increased density is caused by an intraälveolar oedema throughout the affected area; in addition, there is the increased density of the pulmonic markings indicative of dilated vessels and associated interstitial type of congestion. Either of these processes may predominate. If the former element predominates, the affected areas may assume a nodular appearance, in which case a neoplastic (metastatic type) or inflammatory (pneumonic type) process may be simulated. If the latter feature is more pronounced, the roentgen findings are those which are commonly encountered in simple cardiac decompensation with resultant hyper-

vascularization of the lung fields. The accentuated markings usually extend uniformly to the peripheral and basal lung fields. In lungs manifesting azotaemic changes, the peripheral portions of both pulmonary beds, the apices and lower lateral portions of the lower lobes are comparatively clear. The costophrenic sulci are seldom obliterated. If the roentgenographic azotaemic complex remains constant, the prognosis is poor, for when recovery does occur, clearing of the lung fields is rapid. Six illustrative cases are cited by the authors.—*Pulmonary Manifestations of Azotemia, R. A. Rendich, A. H. Levy & A. M. Cove, Am. J. Roentgenol., December, 1941, 46: 802.*—(*W. H. M.*)

Interlobar Empyema.—Sixteen cases of localized interlobar empyema were found among the case records of 650 cases of empyema treated at the Mount Sinai Hospital of New York City in the past ten years. All were found to be due to a ruptured lung abscess. The following findings, one or more of which were present in the 9 cases, point to a definite diagnosis of interlobar disease, or are sufficiently suggestive to warrant a consideration of such a diagnosis: (*1*) an interlobar collection of fluid, air, or both fluid and air, (*2*) a fluid level traversing a fissure or abutting on a fissure, (*3*) an alteration of the shape of a fissure limiting an intrathoracic lesion, (*4*) progression of disease along a fissure. The last three signs are not infallible, as cases have been seen in which these signs were found and in which operation proved the disease to be entirely intrapulmonary. Even a fusiform shadow in the known location of a fissure has been noted in a case in which the disease did not involve the pleura. Only the presence of fluid and air loculated within interlobar pleural surfaces can be regarded as pathognomonic. There are no definite clinical criteria for the recognition of this lesion.—*The Roentgenographic Features of Perforated Interlobar Abscess (Interlobar Empyema), B. Copleman & H. Neuhof, Am. J. Roentgenol., December, 1941, 46: 798.*—(*W. H. M.*)

Haemothorax.—In a series of 86 cases of haemothorax there were 39 cases caused by trauma, 35 caused by primary pulmonary carcinoma, 3 by pulmonary tuberculosis, 2 each by cardiac disease, sarcoma of the ribs and metastatic carcinoma of the lung and one each by Banti's disease, lymphosarcoma and lobar pneumonia. The 35 cases caused by carcinoma constituted 14 per cent of a total of 247 proven cases of primary carcinoma of the lung. The location of the cancer had no definite relation to the presence of haemothorax. Of the 35 cases, 15 had adenocarcinoma, 3 had oatcell carcinoma, one had an endothelioma, one a Pancoast tumor and 12 had carcinomata of an unidentified type. The pleural fluid was studied in 23 cases, 11 of which showed cancer cells. Bronchoscopy was performed in 8 cases, but the fluid revealed cancer cells in only one of these. Bronchoscopy should be done in all cases of haemothorax. Haemothorax appears to be uncommonly associated with metastatic carcinoma. When carcinoma cells are found in the fluid or a malignancy is seen on bronchoscopy, surgical intervention is contraindicated.—*Haemothorax: Its Relation to Primary Carcinoma of the Lung, H. L. Cabitt, J. Thoracic Surg., June, 1941, 10: 590.*—(*D. G. F.*)

Tumor of Trachea.—A case is reported of a twenty-five year old male with a history of persistent catarrh beginning with a severe cold six months previously and associated with whistling sounds in the chest. Attacks of dry cough appeared one week before examination. Once it was followed by haemoptysis. On physical examination there was an increase in the percussion note with diminution of vocal fremitus and breath sounds over the right chest. On X-ray examination a dense shadow was present at the right base and there was a small pneumothorax extending from apex to base. Later examination revealed a gradual decrease in these findings to be followed four months later by emphysema of the entire lower lobe. The succession of atelectasis and emphysema is a sign of pri-

mary importance in the diagnosis of intrabronchial tumor. The atelectasis appears to be caused by the accumulation of bronchial secretion completing the obstruction of a bronchus only partly obstructed by tumor; when this is overcome air exchange is partially reëstablished followed by disappearance of the atelectasis and the appearance of emphysema. Bronchoscopic examination revealed a tumor at the level of the right inferior main bronchus. Following the removal of a piece for biopsy a severe haemoptysis occurred and the patient died the following day. At autopsy a piriform tumor was found at the level of the tracheal bifurcation. Its narrower end extended into the right bronchus and a blood clot obstructed the lumen of this bronchus beyond the tumor. The tumor was composed essentially of multinuclear cells of the myeloplax type; many areas of the tumor appeared mucoid and others appeared fibromatous. A process of ossification was found at the lower border of the terminal tracheal cartilage and, separating from the surface of ossification, a granuloma making up the tumor was seen. This giant cell tumor (polymorphous benign myeloid sarcoma) is thought to be the first of its type described in the trachea. Its rarity is attributed to the fact that the giant cell tumor is a bone tumor and the trachea does not tend to ossify even in advanced age.—*Giant Cell Tumor of the Trachea, J. M. Cid & J. L. Bonilla, Jr., J. Thoracic Surg., December, 1941, 11: 210.*—(*D. G. F.*)

Sarcoma of Bronchus.—The patient whose history is presented was a thirty-two year old woman who was first seen in Bellevue Hospital in 1938. At that time a diagnosis of sarcoma of the bronchus was made and X-ray treatment instituted. In the ensuing years repeated bronchoscopic examination showed that the tumor had practically disappeared. Roentgenograms between March, 1938 and August, 1939 were found to be normal in appearance. In June of 1940 a dry cough developed and at this same time roentgenographic changes were noted in the right upper lobe. In August, 1940 the patient was readmitted to the hospital because of an acute febrile illness which was thought to be a pulmonary abscess developing secondary to a blocked bronchus. However, it was subsequently found that the sputum was positive for acid-fast bacilli. The patient died with an acute exudative tuberculous pneumonia in the left lung and at autopsy extensive caseous tuberculosis was found in both lungs. The only remaining evidence of the previously described sarcoma was a small nodule at the bifurcation of the upper and lower lobe bronchi of the right lung. It is believed that the terminal tuberculous infection was caused by the breaking down of an old tuberculous focus by the extensive roentgen therapy the patient had. The spindle sarcoma of the bronchus was apparently cured by deep X-ray therapy.—*A Case of Spindle Cell Sarcoma of the Bronchus: Supplementary Report, O. S. Baum, G. Silverman, R. Rosh & R. L. Riley, Ann. Int. Med., October, 1942, 17: 750.*—(*A. A. E.*)

Obstruction of Bronchus by Metastatic Carcinoma.—A case is presented of a fifty-nine year old female who had had a resection of the sigmoid for a small obstructive annular carcinoma. About nine months later she developed cough, occasional haemoptysis and moderate fever which persisted for nearly a year. X-ray examination revealed increased density of the right middle and lower lobes near the hilum. On bronchoscopic examination a mass was observed to fill the lumen of the right upper lobe bronchus below its orifice. Biopsy revealed adenocarcinoma. Because of the possibility that this might be primary a right pneumonectomy was done. Convalescense was uneventful. Microscopical examination, however, revealed typical adenocarcinoma similar to that in the colon. At the time of writing she was felt to have metastasis near the liver.—*Metastatic Carcinoma of the Lung Invading and Obstructing the Bronchus, F. Raine, J. Thoracic Surg., December, 1941, 11: 216.*—(*D. G. F.*)

Primary Tumors of Diaphragm.—Primary tumors of the diaphragm are rare. They occur at all ages. Benign tumors such as lipoma, fibroma and one angiofibroma have been reported. The malignant neoplasms, fibrosarcoma, fibromyosarcoma, rhabdomyosarcoma and leiomyosarcoma grow very rapidly and are often large enough to displace the lung and mediastinum. Metastases to the lung, pleura and bones occur frequently. Symptoms in most of the cases are: (*1*) lower chest pain, referred to the hypochondrium, increased on respiration and occasionally radiated along the distribution of the intercostal nerves; (*2*) dyspnoea; (*3*) dry cough. Haemoptysis is less common. The tumor may be palpable in the hypochondrium and moves with respiration. Enlargement of the liver or spleen is apt to be erroneously diagnosed. The diagnosis of diaphragmatic tumor usually cannot be expected without the aid of supplementary methods. Thoracoscopy permits direct visualization of the tumor and its relation to the surrounding structures. Roentgenoscopy is indispensable in determining whether the tumor is of intrathoracic or abdominal origin. Basal intrathoracic tumors show respiratory motion, synchronous with the normal excursion of the diaphragm. If the tumor lies underneath the diaphragm and protrudes through it into the thorax, a paradoxical movement of the tumor shadow is noted, similar to that characteristic of diaphragmatic hernias. The paradoxical movement may not be demonstrable in the presence of adhesions. By means of pneumoperitoneum a circumscribed subphrenic mass can be clearly differentiated from a tumor extending into the thoracic cavity. Furthermore if there is a permeable defect in the diaphragm, a pneumothorax will be produced. The use of artificial pneumothorax usually determines whether a shadow noted at the base is of pulmonary or extrapulmonary origin. The intrapulmonary tumors will usually separate from the diaphragm when a good compression of the lung is obtained. A tumor of the diaphragm or diaphragmatic pleura rests with its base on the diaphragm and is separated from the collapsed lung which shows no pathological changes. Encapsulated effusions at the base are detected by the appearance of shifting fluid levels after injection of air. If a satisfactory pulmonary collapse is prevented by simultaneous involvement of the pleura, the differentiation of the diaphragmatic tumor from pulmonary disease may become impossible. Such a difficulty may be encountered in the case of pulmonary sarcomata which may grow by either infiltration and destruction of the lung or by expansion. The author reports 2 cases in which a diagnosis of diaphragmatic tumor was made. In the first case a chest roentgenogram showed a large, circumscribed shadow resting with a broad base on the dome of the right leaf of the diaphragm, from which it was not separated by interposed pulmonary parenchyma. The upper border of the shadow was polycyclic, suggesting either a lobulated tumor or a mass of multicentric origin. Later roentgenograms showed progressive enlargement of the mass which ultimately extended to the lateral chest wall; the heart was displaced to the left. On roentgenoscopy an anterior and lateral displacement of the oesophagus was demonstrated. Diagnostic pneumothorax revealed fluid at the base. A pneumoperitoneum showed free air underneath the diaphragm. A diagnosis of sarcoma of the diaphragm or diaphragmatic pleura was made. At operation a large fungating tumor was found to arise from the dome of the right leaf of the diaphragm. The histological diagnosis was fibrosarcoma of very malignant grade. In the second case the chest roentgenogram showed a round, sharply defined shadow at the base of the right lung field. This moved with the diaphragm on deep inspiration. On oblique views the shadow localized slightly posteriorly from the dome of the diaphragm from which it could not be separated. Further studies could not be made.—*Primary Tumors of the Diaphragm Roentgenologically Considered, A. J. Ackermann, Am. J. Roentgenol., May, 1942, 47: 711.*—(*W. H. M.*)

Johne's Disease.—Johne's disease is a chronic, infectious disease of cattle and sheep. It is characterized by recurrent diarrhoea and emaciation and is caused by an acid-fast rod. Intracutaneous johnin tests were done in farm animals in Arkansas. The procedure is the same as in intracutaneous tuberculin tests. Horses, mules, cattle, sheep, goats and swine were sensitive to johnin. Acid-fast organisms were found in the mucosa of the intestines in about 50 per cent of reacting cattle, sheep and swine that have been examined postmortem. The infection is transmitted by ingestion of the organisms. Isolation can prevent spread of the disease. It is probable that Johne's disease acts as a predisposing factor in the production of other disease conditions in cattle and sheep.—*Johne's Disease in Farm Animals, D. F. Eveleth & Rebecca Gifford, J. Am. Vet. M. A., January, 1943, **102:** 27.*—(*G. C. L.*)

Key to Abstractors

A. A. E.: Adrian A. Ehler, Albany, New York.
D. G. F.: David G. Freiman, Bedford Hills, New York.
D. H. C.: David H. Cohen, Oteen, North Carolina.
G. C. L.: George C. Leiner, New York, New York.
G. L. L.: George L. Leslie, Fort Jackson, South Carolina.
H. M.: Henriette Marcus, Biloxi, Mississippi.
J. B.: Joseph Ballinger, New York, New York.
W. H. M.: W. H. Merrit, New York, New York.